

Forever Fiber

Dietary fiber came into its own more than two decades ago. Unlike other dietary fads, however, research has continued to bear out the fact that fiber plays an important role in health promotion.

In the early 1970s, dietary fiber was the hottest new prospect on the nutrition scene, and enthusiasts believed that a lack of fiber could explain every ill that plagues the Western world, from constipation to cancer. An advantage of these fads is that they generate much new research. However, the facts established by such research often fail to fulfill the initial high hopes, and within a few years the field is largely abandoned and media attention shifts to the next magic bullet. This is what happened to dietary fiber—but some researchers doggedly hang on, sometimes with unexpected results. The recent report from the Health Professionals Study¹ confirms that the field of dietary fiber is still alive and kicking.

The Health Professionals Study is the most recent of three large prospective, observational studies conducted by Dr. Walter Willett and colleagues at Harvard University; the other two are the Nurses' Health Study and the Physicians' Health Study. An essential tool in all of these studies is a computer-readable food frequency questionnaire. By the start of the Health Professionals Study in 1986, this questionnaire had expanded to 131 food items. Comparisons with biomarkers and diet records show it to be about as valid as traditional, more laborious methods for assessing dietary intakes.

Previous accounts of the Health Professionals cohort have reported protective effects on coronary risk of vitamin E and alcohol and little or no effect of iron or coffee intake; effects of fish consumption were also limited.² Rimm et al.¹ now report that men who ate more fiber, especially fiber from cereals, suffered fewer heart attacks. The paper is clear and well written, provides all the relevant facts, and methodically discusses most of the objections that might be raised.

The major weakness of any study based on observational epidemiology is the risk of confounding. This is not as relevant when relative risks are high, such as the 10–20-fold increase in lung cancer risk seen in smokers. However, when relative risks are less than three to four, there is always a nagging

question as to whether the factor ostensibly associated with disease could be a surrogate for some other real cause that was not measured with sufficient precision or not measured at all. In many studies a variety of healthy behaviors tend to occur simultaneously in the same subjects. The present report is no exception: men in the highest one-fifth of fiber intake are also leaner, smoke less, are more active, take more vitamin E supplements, and eat less fat and cholesterol than the average participants.¹ Previous reports³ have suggested that they also eat more fish and less red meat.

This report, and previous ones as well, prove beyond reasonable doubt that this package of healthy behaviors together offers some protection against heart disease; the challenge is to find out which components are responsible. Previous analyses of diet and heart disease in the Health Professionals Study suggested that vitamin E pills conferred protection—a suggestion partly borne out by a recent randomized trial,⁴ as did regular moderate alcohol intake. The authors used multivariate analyses to adjust for these and other factors known or suspected to promote or protect against coronary heart disease, including smoking, self-reported hypercholesterolemia or hypertension, obesity, physical activity, and a number of dietary factors. As a result, the protective effect of fiber was somewhat diminished—the relative risk in men in the highest fifth, with an intake of 29 g per day relative to men in the lowest fifth, who ate 12.4 g per day, increased from 0.59 before to 0.64 after adjustment for known risk factors.

On the one hand, such an attenuation of risk is reason for concern. The confounding variables as measured are always a less-than-perfect reflection of the true long-term behavior of each subject. The more unreliable the estimate of certain variables (e.g., smoking) becomes, the more the confounding effect of that variable is underestimated. On the other hand, when low fiber intake coincides with known risk factors, one can hardly expect that the calculated risk will remain unchanged after multivariate adjustment for these factors. In the present case, adjustment for the full package of known confounders changed the risk reduction from 41% to 36%. The authors do not provide calculations of what change should have been expected given the known effects of the risk factors in question on coronary disease plus the imperfections in the way they were measured. However, the attenuation was only modest, which leaves plenty of room for a real effect of fiber.

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However, other issues also need to be addressed before one accepts that fiber protects against heart disease. Could the inverse relation between fiber intake and incidence of coronary disease be an artifact caused by imperfections in the dietary assessment method? This is an argument often raised in connection with the Willett food frequency questionnaire. I do not believe this argument holds. Random errors in the assessment of food intake will attenuate rather than increase the strength of any association between diet and disease and result in a relative risk closer to 1 and a risk reduction closer to 0%. The authors actually quantitated the effect of measurement error and suggest that reduction of these errors to zero would have increased the strength of the association and produced a relative risk of 0.56 instead of the 0.64 seen.

Of course, "random noise" will also occasionally produce a spuriously high correlation if enough relations are tested; correlating the intake of hundreds of nutrients with heart disease rates would produce dozens of "significant" associations. However, the present study of fiber was not such a fishing expedition: the hypothesis that dietary fiber reduces heart disease risk was first proposed by Cleave, Burkitt, and Trowell in the 1960s⁵ and has been confirmed in other epidemiologic studies.⁶

The problem with accepting a link between fiber intake and heart disease has been not so much a lack of consistency in the epidemiologic data but rather the problem of confounding, plus the lack of an explanation of how fiber should work. Explaining the association would be easier if only the soluble fiber found in oats, fruits, and vegetables were associated with less risk, because soluble fiber lowers cholesterol.^{7,8} However, in the present study, the insoluble fiber from wheat rather than soluble fiber was associated with less coronary disease. This type of fiber is not known to lower cholesterol⁹ and may even raise it.¹⁰ Rimm et al.¹ evoke the long-term impact of a diet high in insoluble fiber on glucose control, plasma lipids, and hemostasis in an attempt to explain their findings. Although this conjecture is difficult to refute, it remains a conjecture.

The only solution to the uncertainty concerning fiber and heart disease is to initiate a long-term clinical trial. In the field of nutrition such trials are often considered unfeasible because of their costs. However, the problem is not so much one of costs as of recovering costs. No commercial company can afford to mount a large clinical trial of fiber and heart

disease because, unlike a drug, whole-wheat bread cannot be patented. Adding a 0.5 US cent to the price of every loaf of bread sold in the United States and Europe could finance enough trials to answer all major questions about cereal fiber and disease, but no government is willing to make itself unpopular by raising taxes on foods for the purpose of financing research. Therefore, we will have to do with whatever we can learn from observational studies such as the Health Professionals Study and eat our bran in the hope that it will do some good. If nothing else, it will reduce constipation, a good enough reason by itself.

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